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# DISEASES of the CHEST

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# DISEASES OF THE CHEST

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C. M. HENDRICKS, EDITOR-IN-CHIEF

(A MONTHLY PUBLICATION)

***"The most important factor in diagnosis in the majority of cases of pulmonary tuberculosis is keeping the disease in mind."***
**Lawrason Brown, M. D.**

## Editorial Comment

**Important Points in Prognosis**

BY CONSIDERATION of the following points, a probable or doubtful valuation of the prognosis in individual cases of pulmonary tuberculosis may be made.

*Heredity.* The importance of this has been much over-estimated.

*Constitutional Factors.* An appraisal of these should be made.

*Mental Character.* A phlegmatic person has advantages over one of liable temperament. Psychic shocks may have a very unfavorable effect. The toxaemic condition due to tuberculosis may itself influence the patient's character.

*The Patient's Age.* The effects of dissemination are better resisted by older than younger subjects.

*Rapidity of Development* of the tuberculous changes, and the results of physical examinations of the lungs.

*Examination of Sputum.* Character and amount. The number and type of bacilli found.

*The Vital Resistance.* May be estimated by careful observation.

*Roentgen Examination.* Extent of lung markings. The smallness of the heart and main arteries, etc.

*General Nutrition.* General appearance, obvious wasting, etc.

*Blood Pressure.* A systolic blood pressure of below 100 mm. Hg. in men and 90 mm. Hg. in women is unfavorable, though the blood pressure may of course, in some cases, be favorably influenced by treatment.

*Haemoptysis.* The danger of this may be increased by narcotics.

*The Urine.* A Diazo reaction may disappear under treatment.

*Complications.* Enlargement of the liver, diarrhea of various causes, anal fistula, thyroid enlargement (possibly a favorable symptom), amyloidosis (the appearance of the tongue may be suggestive in that respect), and laryngeal tuberculosis.

*Signs* of vagotonic or sympathico-tonic tendency.

*Pyrexia.* And various other toxic signs.

*Data* furnished by modern clinical laboratory examinations.

*The Influence* of other diseases such as syphilis and diabetis mellitus. C.M.H.

**The Incidence of Intestinal Tuberculosis**

RECENT REPORTS on autopsies on patients who had pulmonary tuberculosis furnish the information that lesions in the intestines are not uncommon. In fact, some have reported finding evidence of the trouble in 60% of the cases autopsied. This, of course, does not mean that such a large percent of those having pulmonary tuberculosis suffer with the disease in the intestines, but it does mean that this complication is often unrecognized and, consequently, untreated.

Many times a diagnosis of this condition is delayed for the reason that we

wait for the two most common symptoms, namely abdominal pain and diarrhea. These are the symptoms of an advanced stage of the disease. We should at least look with suspicion upon such early symptoms as aversion to food, the frequency of intestinal flatus, mild attacks of nausea, and the uncomfortably "sick feeling" of which many patients complain, but cannot clearly describe. R. B. H.

**Laryngeal Tuberculosis** EARLY DIAGNOSIS is essential to the successful treatment of this disease. Routine laryngologic examinations should be made in every case of pulmonary tuberculosis, since tuberculosis of the larynx is a common complication. This condition has not received the attention that it deserves because of the prevalent idea among general practitioners that little or nothing can be done to relieve the condition. In all cases of chronic laryngitis which do not respond readily to the removal of the etiologic factor, such as sinus infection or mis-use of the voice, a careful study should be made to exclude pulmonary tuberculosis.

Patients with a definitely diagnosed case of tuberculosis of the larynx are best handled in a sanatorium for tuberculosis, where they are under constant observation. A disease of the larynx superimposed on a severe pulmonary infection should be accepted as tuberculosis of the larynx until proved otherwise. C.M.H.

**Our Crusade** THE REAL CRUSADE against tuberculosis, conceived in recent years as pivoting on the question of the open case, must be conceded to be a most tremendous force in the control of the disease. The crusade which aims at the breaking of contact between the tuberculous individual and the child, must be regarded as most efficacious. Unfortunately, in most communities, this campaign is not yet pursued with sufficient energy or consistency. The pursuance of this policy will necessarily test the tact, efficiency and humanitarian insight of the officers of any state, county or municipal institution. The fulfillment of this policy, however, from the standpoint of efficiency in the prevention of infection is worth every effort and every sacrifice.

The contact program in every tuberculosis institution, especially state controlled, should receive minute and detailed attention. Other things being equal, our future campaign must give the open case, in contact with children, preference as regards sanatorium admission. There can be no other procedure countenanced in public institutions. C.M.H.

**In Considering the Death Rate** IN THE consideration of the rather sharp decline in the death rate from tuberculosis, not only in the United States but throughout the world, there are many questions we should ask ourselves before claiming this or that movement responsible, viz.:

*How much credit must be given the normal cycles in the tuberculosis death rate?* That there are cycles is proved beyond doubt.

*How much credit must we give to the world war?* The great increase during the world war of the mortality rate throughout the world must be construed as an increase in the number of deaths of individuals already suffering from tuberculosis. Privation, lack of care, mental anguish during the period of the war, no doubt brought about this increase in the number of deaths; as a consequence, the elimination of these individuals would tend to decrease the death rate on the restoration of normal conditions.

*How much credit must be given the tuberculosis crusade?* Here we must recognize the good that has been attained by segregation of open cases; the publicity given hygiene, etc.

*How much credit must be given to the better standard of living?*

*How much credit must be given to the complicated process of immunity?*

*How much credit must be given to the higher standard of education?*

*How much credit must be given to certain methods of treatment that have prolonged the lives of tuberculous individuals?*

*How much credit must be attributed to the world-wide establishment of sanatoria?*

*How much credit must be given the great influenza epidemic that carried off great numbers of tuberculous individuals, as well as individuals who were either predisposed or had they lived inevitably would have become tuberculous?*

At the present time one can truly say that the combination of the above factors has brought about this decline. It is impossible to evaluate each individual factor mentioned; however, it is the opinion of many that the outstanding factors are as follows: the cycle, the world war, the influenza epidemic, and increased efforts toward the segregation of the open case. C.M.H.

**Great Events and Medical Progress** GREAT EVENTS in history are proved to be landmarks in the progress of medicine and civilization.

The Crusades brought to western Europe Arabic pharmacy, which influenced a great change in the treatment of diseases. So important became the pharmaceuticals of the Orient at that time that men were driven to seek cheaper methods of transportation and set out in ships to seek short routes to the Orient. As a result Columbus discovered America.

When Constantinople fell western Europe was again blessed, because the great scholars of that city fled to western Europe and their influence for good gave a new stimulus to greater progress of medicine.

Following the invention of the printing press, self-education began, and independent thought soon laid the basis for scientific medicine.

The World War brought about great progress in surgery, the most notable of which, the last division of surgery, thoracic surgery, has been created. Prior to the World War, surgical operations on the thorax, with a few notable exceptions, were confined to resection of the ribs, artificial pneumothorax, or the occasional successful suture of the heart muscles following a knife wound. The great number of chest injuries and the wide prevalence of empyema following the pneumonia prevailing during the period of the war made it possible and desirable to perfect means by which these conditions could be met. Rapid and brilliant have been the achievements in this field, so that at present the medical man has at his disposal the surgical relief of chest conditions which formerly were primarily medical problems.

The progress of chest surgery has become so great that the thorax is now as accessible to the surgeon as the abdomen. C.M.H.

**Prognosis of Cavities** THE PROGNOSIS of cavities not given suitable therapy is very bad indeed. Eighty per cent of such patients will succumb within three or four years. The majority of cavities continue to get larger by appositional growth, caseation and softening of the wall of granulation. The danger is that of dissemination, throughout the adjacent or remote parts of the lung, by aspiration. Another great danger is that of hemorrhage; fatal bleeding is much more frequent than supposed. The spread of the tuberculous process to the larynx and intestine by inoculation is especially dangerous.

Collapse therapy is the only method of choice in treating cavities. It should not be undertaken without a precise anatomic diagnosis. Of greatest importance is the condition of the sounder lung, especially the finding of small foci of disintegration. The situation of the cavity is of great importance if thoracoplasty is contemplated.

Spontaneous cure of cavities is limited to early cavities and occurs only rarely. Collapse therapy, it is estimated, will cure early cavities in about seventy percent of the cases and in about fifty percent of the older cavities. C.M.H.

# Papworth Village Settlement;* Its History and Aims

BY
SIR PENDRILL VARRIER-JONES
F.R.C.P.
*Cambridge, England*

I FEEL very privileged to be asked to write an article on Papworth Village Settlement for DISEASES OF THE CHEST. For many years I was a voice crying in the wilderness, and it seemed that few heard, and still fewer wanted to hear. It is therefore a particular pleasure to contribute to such a magazine as this.

Papworth is simply an expression of medical thought in material form. We have for years told consumptives what to do after leaving the sanatorium. At Papworth we have gone a little further. We have enabled them to do it.

In Great Britain there is no such thing as a *light* open-air job. Most open-air jobs are heavy; and such light jobs as there are anywhere usually mean a light pay envelope. Light pay envelopes mean a low standard of living; and a low standard of living means disaster for the patient and danger for his family and all his "contacts."

It seemed to me, in 1914, that it was absurd to tell patients to obtain work which did not exist and to live in conditions far beyond their means; and it then occurred to me that no progress would be made unless and until suitable work and conditions were provided. If the advice I tendered was sound, it ought to be carried out; and, if carried out, it ought to produce highly satisfactory results.

In 1914, therefore, I began with one patient. Others came into the scheme; and in time workshops were built and arrangements were made for suitable patients, after sanatorium treatment was over, to find permanent paid employment in industries of their own creation.

In 1918 we purchased Papworth Hall and its grounds. At that time we had 25 patients, living in the Hall itself. As their active treatment concluded, some decided to take the chance offered to them and to live and work in Papworth's new industries. In that year we sold goods worth $1,950 and paid wages amounting to $845.

The scheme proved popular. It grew. As it increased in scale more and more patients were admitted to the hospital section for treatment, and more ex-patients became "settlers;" i. e., brought their families to live at Papworth and took jobs in the Industries.

By the end of 1927 we had 200 beds available for patients under treatment, and we had admitted women to the benefits of the scheme. The industries by this time were paying nearly $90,000 in wages, and selling some $230,000 of goods each year without any appreciable loss; and the scheme began to attract attention. At first people were surprised to find that consumptives were willing to settle in a "colony." Then they were affronted at the idea of the families being "settled" too, not realizing that the family is the unit with which—for obvious economic and sociological reasons—any comprehensive anti-tuberculosis scheme must deal. We were told on good authority that the children born in the village would die, almost at birth, from tuberculous meningitis; and we were also told by other authorities that they would live until they grew up and left Papworth, but would then be stricken down at once by the disease. The truth is that neither disaster occurs. The children simply do not get tuberculosis, whether they stay at Papworth, or leave. Why is this? Surely because, living as they do with a "settler" who is a "middle" case and therefore from time to time a source of infection, they get small doses which are not enough to cause disease since (a) resistance is high, thanks to good food and healthy conditions, and (b) the specially built cottage and the habit of keeping the windows

*Near Cambridge, England.

open prevent mass doses. We cannot yet claim to have systematized immunity; but are we not, perhaps, providing some helpful indications?

Progress did not stop in 1927, though by that time we were receiving less advice than in our early years, and Papworth continued to grow though nothing like as fast as conditions required. All development is limited by two factors: the charitable income and the volume of sales. We have found that if we can obtain the capital free of charge the scheme supports itself, so long as the sales department can find a market for the goods manufactured. We can only get capital, *free*, by means of charity drives and we wish we had the benefit of your Christmas Seals, and we can only get sales by making the right goods and selling them at the right price.

At the end of 1934 our entire capital investment did not greatly exceed a million dollars and we had over 1,000 people in the village. Sales surpassed $410,000 and wages totalled $150,000. In a sense we were paying, in wages, just about 15% on our total capital: and when it is remembered how much our employees would have drawn in unemployment and health benefit had we not employed them the cash value of Papworth can be approximately assessed.

In this calculation it must not be forgotten that our main capital investment is not in the industrial departments at all. It is in hospital buildings, laboratories, land, and, above all, houses. We have now three hospitals, with a surgical section for cases requiring thoracic surgery now being added; a sanatorium section composed of chalets; three hostels; and an Out-patient department for the care of the village population. We have accommodation for 446 patients, and a waiting list. In the industries we have eleven departments. We do our own building and printing; we keep poultry, we make travelling goods, furniture, and portable wooden buildings. We carry on a trade in upholstery and we write signs. In every case each department has grown up round a skilled ex-patient, and the whole of the industries is managed by an ex-patient as well.

Such, very briefly, is Papworth. Its aim is to become a really comprehensive and complete unit capable of benefiting any sufferer from any sort of chest disease; to provide both medical and surgical treatment; and to provide employment for those who, while not bedridden, are nevertheless too ill to stand the pace and strain of modern industry. Further, we recognize that we have almost if not quite unique opportunities for research: for our system of permanent employment gives us the chance of observing cases and their contacts closely and continuously for years at a stretch.

In conclusion, may I suggest the main lines upon which our minds are now working? We believe that little or no further progress will be made in the conquest of tuberculosis by the old dispensary-sanatorium system, not because that system is wrong, but because it is incomplete. It does not attract early cases, because it provides so little "bait," in the form of cures, to encourage them to submit themselves for treatment. Two-thirds of the beds in British sanatoria are therefore filled by middle and advanced cases who are beyond cure and can hope only for quiescence; and so long as we content ourselves by producing quiescence in the sanatorium, and then returning patients to their homes armed only with advice which they cannot follow, we shall continue to frighten off suspects who might otherwise come forward early for treatment.

We believe, too, that prevention is better than cure. We suspect that pathology may have taught us all that it can, and that perhaps that teaching has not been of much real help. We are inclined to think that we ought to address ourselves to the study of the chemical changes which must precede tissue destruction, and to reduce diathesis to bio-chemical terms. May there not be some system by which one may assess the individual degree of resistance to various diseases, and ap-

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# Intrapulmonary Infection of Bronchogenic Origin*

BY
CARL J. JOHANNESSON, M.D.
*Walla Walla, Wash.*

BRONCHOGENIC EXTENSION of intrapulmonary infection from one lung across to the other has been observed frequently in the routine examination of roentgen films during my experiences at various hospitals. Such extension of infection from one lung, in which destruction of pulmonary tissue has taken place, gives rise to infection in the opposite lung and is no doubt an autogenous infection, due apparently to the inhalation of infectious exudate and detritus, as it is most frequently observed in the presence of a breaking-down or cavitation of a caseous tuberculous lesion, though it may also occur from other intrapulmonary suppurations and destructive lesions.

The studies carried out in Krause's laboratories in Baltimore (1) have shown the presence in the walls of the larger bronchi of small nests or pockets of lymphatic tissue, covered only by a thin layer of bronchial mucosa. Krause believes that these islands of lymphatic tissue drain the bronchial mucosa, that tubercle bacilli which invade them are carried thence through lymphatic channels into the thoracic duct which in turn conveys them to the venous blood stream in the neck, whence they ultimately find lodgment in the pulmonary tissue by way of the pulmonary artery. It is quite conceivable that pyogenic organisms also find their way into these islands of lymphatic tissue, whence they, too, may reach the lung by way of the pulmonary artery. These small islands of lymphatic tissue may also be invaded by a more virulent organism which destroys the lymphatic tissue and the contagious pulmonary tissue, resulting in suppuration and destruction. The destruction of these pockets of lymphatic tissue by a bronchogenic spread or extension of the infection could well result in multiple saccular dilatations of the bronchi or honeycombed cavitations.

Whatever may be the part played by these islands of lymphatic tissue, there can be little doubt of the bronchogenic origin of intrapulmonary suppuration, as demonstrated by the important experimental work of Smith (2), Crowe (3), Scarf (4), Allen (5), Myerson (6), Van Allen (7), Hill (8), Miller (9), and others.

When the destructive lesion involves particularly the right upper lobe (fig. 1), one would expect aspiration infection of the right lower lobe to precede bronchogenic cross-infection, as the bronchus to the right lower lobe is an almost direct continuation of the trachea. This type of secondary infection does occur, of course, quite frequently, usually in association with bronchogenic extension, but the infection is largely limited to the peribronchial tissue and those alveoli adjoining the bronchi. The secondary infection in this region does not as a rule involve the peripheral parenchyma to the extent that it is involved in bronchogenic cross-infection.

Bronchogenic extension in cross-infection may occur in any part of the opposite lung, but in reviewing a large series of roentgen films, made in the usual upright posteroanterior stereoscopic exposures, the area involved is between the second and

*Reprint from Northwest Medicine, Seattle, Vol. 34, No. 5, Page 176, May, 1935.

1. Krause, A. K.: Experimental Studies on Tuberculous Infection. The Harvey Lectures, 1921-22. J. B. Lippincott Co., New York, 1923.
2. Smith, D. T.: Experimental Aspiratory Abscess. Arch. Surg., 14:231-239 (part 2), Jan., 1927.
3. Crowe, S. J., and Scarff, J. E.: Experimental Abscess of Lung in Dog. Arch. Surg., 16:176-178 (part 2), Jan., 1928.
4. Scarff, J. E.: Experimental Production of Pulmonary Abscess; Etiologic Factors. Arch. Surg., 18:1960-1983, April, 1929.
5. Allen, D. S.: Etiology of Abscess of Lung; Experimental and Clinical Studies. Arch. Surg., 16:179-191, (part 2), Jan. 1928.
6. Myerson, M. C.: Pulmonary Aspects of Tonsillectomy Under General Anesthesia. Laryngoscope, 32:929-942, Dec., 1922.
7. Van Allen, C. M., and Adams, W. E.: The Effect of Bronchogenic Contamination in Embolic Lung Lesions. Tr. Am. A. Thoracic Surg. (to be published).
8. Hill, E. C.: Radiopaque Bismuth Suspension for Anatomical, Histological and Pathological Research. Bull. Johns Hopkins Hosp., 44:248-265, April, 1929.
9. Miller, W. S.: Arrangement of Bronchial Vessels. Anat. Anz. Jena., 28:432-436, 1906.

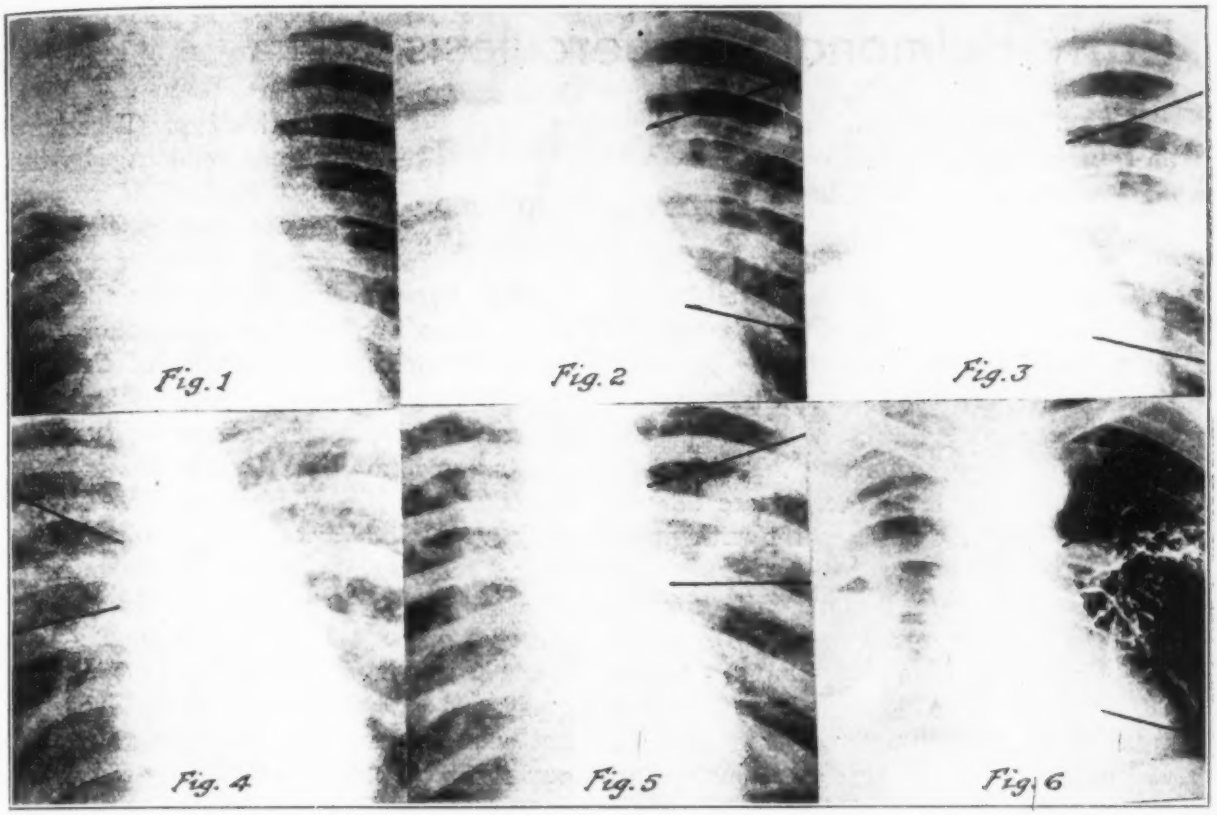


Fig. 1, Case 1. Feb. 28. 1933. Diffuse infiltration with almost complete excavation of right upper lobe. At this time there is no secondary infection into the left lung.

Fig. 2, Case 1. June 20, 1933. Partial collapse of right lung has been performed. At this time the area of bronchogenic cross-infection was first recognized in the left lung; note site of predilection opposite second to fourth ribs.

Fig. 3, Case 1. Sept. 1, 1933. Shows progressive involvement of the right lung. At this time very extensive involvement of the left lung is seen; still remaining in region of site of predilection.

Fig. 4, Case 2. May 5, 1930. Tuberculosis of left lung with presence of small cavitation. Bronchogenic extension of infection is seen in the right lung opposite the second to fourth ribs.

Fig. 5, Case 3. Nov. 28, 1932. Tuberculosis of right upper lobe. Secondary or bronchogenic cross-infection of the left lung. This condition is most frequently seen.

Fig. 6, Case 4. April 2, 1930. "Bronchial spill" or artificially demonstrated site of predilection of intrapulmonary material from the right lung into the middle of the left lung by gravity. Patient lying on left side and lipiodol injected through drainage tubes into right chest. Through a bronchial fistula the lipiodol reached the main right bronchus, gravitated around the bronchial bifurcation into left bronchus and out into the parenchymatous tissue opposite the second to fourth ribs.

fourth ribs. In the early stage there will usually be found a limited focus of infection somewhere in this area (fig. 2), while later the entire area will be involved and extending out to the periphery (fig. 3).

Most frequently the original infection is found in the right upper lobe and spreads to the left lung, but figure 4 shows an example of the original infection being in the left upper lobe with the bronchogenic secondary infection into the right lung, and the site of predilection being relatively the same.

In the ordinary type of subacute pulmonary tuberculosis of the adult the lung apices of both upper lobes are very frequently involved. The lesions in the two lungs may be relatively of the same age and distribution, or the lesion on one side may be very apparently of longer duration and more widespread than that on the opposite side.

When the lesions are of different ages it is generally impossible to determine whether the younger infection is exogenous or an example of autogenous bronchogenic extension. The usual conception has always been that these are examples of autogenous bronchogenic cross-infection.

In all the cases of destructive pulmonary lesions which the author has followed closely, cross-infection has always developed in the part of the lung fields projected between the second and fourth ribs on the roentgen films (fig. 5). The evidence would, therefore, indicate that bronchogenic extension of infection to the apical region of the opposite lung is very unusual.

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# Early Pulmonary Tuberculosis

BY
S. C. DAVIS, M.D.*
*and*
REDFORD A. WILSON, M.D.*
*Tucson, Arizona*

WHEN WE READ statistics on the deaths due to tuberculosis, we are forced, in the words of Will Rogers, "to point with pride and view with alarm." It is a well-known fact that the deaths due to tuberculosis have been reduced greatly in the last thirty years. In the United States registration area in 1900 there were 182 deaths per 100,000 pupulation due to pulmonary tuberculosis; in 1929 there were 68 per 100,000. We may justly point with pride at these figures but of greater importance is the fact that the majority of tuberculosis occurs in the age period 15 to 45, the period of a man's or woman's greatest economic value. In the registration area, more deaths between the ages of 15 and 35 were due to tuberculosis than to any other disease. Furthermore, it is estimated that there are more than 700,000 cases of active pulmonary tuberculosis in the United States today. Until we can reduce this devastating scourge of young adults, anything tending to help in its control should be given our attention and receive our most serious consideration.

It would be superfluous to go into detail regarding the necessity of early diagnosis but this is the most important single factor in saving these young adults from a long and serious illness and possible death. If we were able to diagnose properly and treat pulmonary tuberculosis in its early stages, our percentage of arrested cases and cures would grow tremendously. Mistakes in diagnosis on the part of the physician are responsible for all too few of these advanced cases. Many, many times the physician does not see the case until the disease is advanced. The onset has been insidious, the patient has not observed, or has not considered important, the early symptoms and has, therefore, not consulted his doctor until the disease has progressed beyond the minimal stage. There are numerous reasons why the patient himself is responsible for not getting his diagnosis made when his chances for cure are at the best; but the lay persons are being educated more and more about early symptoms of the disease through insurance companies' and pharmaceutical manufacturers' advertisements, syndicated health columns in daily papers, radio talks, Hygeia, etc. This is very valuable work along public health lines. It promises better health and increases longevity by making the patient more cognizant of the meaning of symptoms and causes him to have more frequent and earlier examinations. Still it is not enough; we, as physicians, must take our share of blame also.

Here lies the crux of the problem—how often and why do we overlook the presence of early pulmonary tuberculosis?

It has been said that a good syphilologist considers that everyone has syphilis until it has been disproven. Likewise, every doctor should consider the possibility of tuberculosis in all patients examined. Many diagnoses of pulmonary tuberculosis are missed because the attending physician was not suspecting nor forever looking for tuberculosis. The doctor doing general practice does not have occasion to see tuberculosis so often as the specialist in chest diseases. However, he should become equally "tuberculosis-minded;" then an early case would be less likely to escape his notice. Therefore, our first plea is for the practitioners to keep the possibility of this disease always prominent in their minds.

The time-worn subject of history-taking may be relegated to our medical school days, but not until we have mentioned a truism, "one case of tuberculosis begets another." History of tuberculous contacts may often save serious blunders, and when such a history exists, tuberculosis must be considered always as a probability.

*Thomas-Davis Clinic, Tucson, Arizona.

The classical symptoms of early tuberculosis are familiar, but so few patients present themselves with these classical symptoms. It is the type without such symptoms which should receive our utmost care, lest an error be made.

We are all too prone to think of tuberculosis as a chronic, slowly progressive disease from its onset. We sometimes forget that it may start acutely, so acutely in fact that it may be mistaken for pneumonia. Indeed, it may start as pneumonia, tuberculous pneumonia, simulating a pneumococci disease, and any case of lobar pneumonia which lasts longer than one ordinarily expects should be studied to rule out the possibility of tuberculosis.

Probably the most deceptive type with which we have to deal is that one which begins acutely with chills, fever, aching, malaise, and is diagnosed influenza. In due time the acute symptoms subside, but the patient is not entirely well. In these cases, tuberculosis must be ruled out by every means at our disposal before that patient is dismissed as non-tuberculous. Well over 50% of all patients coming into the Southern Pacific Tuberculosis Sanitarium, give a history of influenza, from which they did not entirely recover, several months prior to the so-called onset of illness. We contend that they did not have influenza—they had pulmonary tuberculosis from the start and a diagnosis made at the onset would save many, many months of illness and would frequently save lives. Tuberculosis begins acutely as often as it begins insidiously and it should always be suspected in any acute, vague illness.

A patient with an unexplained pulmonary hemorrhage deserves everything in our diagnostic armamentarium to prove or disprove the presence of tuberculous lesion. Many patients have a hemorrhage very early. This often occurs before the history or physical signs will point to a pulmonary lesion. It is our firm conviction that any patient with a pulmonary hemorrhage should be considered as tuberculous. The burden of proof is on the person who feels that the hemorrhage was not due to tuberculosis and, until such definite proof can be produced, we feel that it should invariably be assumed that it is tuberculosis and appropriate treatment be instituted immediately, even in the absence of other signs and symptoms of tuberculosis.

Keeping tuberculosis foremost in our minds, careful history-taking and careful physical examinations are not enough to avoid missing some of these early cases of pulmonary tuberculosis. Our laboratories must be used freely. They may prove a diagnosis in which physical signs are absent. Sputum, all sputum the patient raises, should be examined daily for many days before we may say the sputum does not contain tubercle bacilli. Even then we cannot say that the sputum is negative. When a positive sputum is suspected and has not been obtained by the usual methods, they may be found frequently by injecting the sputum into a guinea pig. If not found by these procedures, then, and only then, can a patient's sputum be said to contain no tubercle bacilli.

The greatest single agent in the diagnosis of early tuberculosis is the Roentgenogram. As compared to the importance of this film in detecting the presence of minimal lesions, the history, the physical signs and other laboratory procedures fade into relative insignificance. Physical signs may be absent or misleading and the diagnosis completely missed without X-ray examination. We admit that we have, on several occasions, had patients with all evidence of pulmonary tuberculosis, including hemorrhage and positive sputum, examinations of whose chests revealed no abnormalities, but whose lesions were found unmistakably on the X-ray film. Hence, we know that the diagnosis of tuberculosis can and will be overlooked often if we depend on physical signs without the aid of the X-ray. When, and only when, we have wider and more extensive use of the Roentgen-ray in patients in whom we have even the least reason to suspect tuberculosis, we

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# Silicosis and Tuberculo-Silicosis*

BY
FRANK PORTER MILLER, M.D.
*Los Angeles, California*

THE DISEASE, Silicosis, is not particularly new as Paracelsus as early as 1534 published his work, entitled "Miner's Diseases." Denker provided the anatomical and Kussmaul the chemical proof of the deposit of inhaled dust, especially silica in the lung. The name pneumonoconosis was invented by Denker, who described in detail the anatomical picture of the dusty lung.

In the early study of silicosis along the Witwatersrand gold fields in South Africa, it brought to light the interesting fact that the majority of miners affected were rock drill workers. The same holds true today whether the mining be gold or otherwise, as the dust exposure is here very excessive. During the period around 1900 ventilation in the mines was very inadequate and the men were working in high temperatures and in relatively high humidity. Dry mining was carried on extensively and at that time the necessity of wet mining was not sufficiently impressed upon the minds of the ultra-conservative miner, and these individuals disliked any innovations in the traditional ways of work, with the result that any suggestion attempting to minimize this occupational danger brought forth only a half-hearted response on their part.

### *Etiology*

Phthisis-producing dust concerns us mainly, and this must possess certain definite characteristics. It must be capable of overcoming the lung's reactive ability to rid itself of a foreign body, and it must remain arrested within the alveoli over a great enough period, and must be relatively indestructible to tissue and tissue juices. Dust becomes arrested by phagocytosis and one must presume that dust particles approximate the size of the common pathogenic micro-organism.

Mavrogordata (1) points out that phthisis producing dust is inert—that it fails to produce like cells that take it up through phagocytosis, and that it fails to provoke exudation. An irritant that produces much expectoration is not retained long within the normal lung. The dust which we are most interested in is the one producing simple silicosis and later tuberculo-silicosis, and the offending agent is silicon dioxide ($SiO_2$). Haldane, many years ago, suggested the possibility that silica which is productive of dust phthisis is often associated with other dusts, which act as an antidote. Certain industries in which silica is present in comparatively large quantities fail to produce simple silicosis—while in others fibrosis begins after relatively short exposure. The problem of dust antidote is receiving considerable attention by those interested in the silicosis problem.

Clinically, there is a factor related to etiology which is unequivocally a dominant one but its explanation upon a physiologic basis is as yet obscure. That is, the super-imposition of an inter-current infection in cases which have remained in a latent stage, ofttimes spells ruin. How these phagocytic cells laden with silica can so obscure themselves until their media is changed through infection has yet to be brought to light.

Mavrogordata is inclined to the view that these cells remain in the lymph of obstructed lymphatics, and lymph will remain as fluid for a long period in stagnant obstructed areas. The progression of disease to exitus in some of my own cases following infections, some of a non-tuberculous nature, were too rapid for comfort.

### *Pathology*

In the development of simple silicosis Strachan and Simpson (2) point out the course of inhaled dust, and their contribution is most noteworthy. Fifty percent of all dust inhaled remains in the nasal mucous membrane. A certain proportion reaches the bronchi and then the ciliated epithelium will wave a great portion of

*Read before the General Medicine Section of the California Medical Association at the sixty-third annual session, Riverside, April 30—May 3, 1934.

this up until it reaches the larynx, and then it is either swallowed or expectorated. Only 4 to 24 per cent of the dust inhaled actually reaches the lung. Deposition of dust upon reaching the lungs, occurs in certain definite sites, and these correspond to the lymphatic arrangement. For our purpose we can consider the pulmonary lymphatic system to consist of a superficial and deep network which is inter-communicating and which empties into either broncho-pulmonary, superior or inferior tracho-bronchial or para-tracheal glands. Furthermore, lymphoid aggregations occur throughout the lung in the sites of the sub-pleural, peri-bronchial, peri-arterial, and peri-venous systems. Early pigmentation will show in the glands, sub-pleural tissue and inter-lobular septa.

Defensive mechanism which attempts to limit the invasion of dust, is the nose and naso-pharynx, reinforced by the ciliated epithelium in the upper respiratory tract. An upper respiratory infection which provokes desquamation of the epithelium removes the greatest obstruction to the particulate matter. When dust reaches the alveoli it produces a proliferation of the lining epithelial cells, which become detached from the walls to form the alveolar phagocytes. The phagocytes or dust cells tend to accumulate in smaller air passages such as the smaller bronchioles, and these pass into the lymphoid aggregates. With the arrest of the phagocytes in the pulmonary lymphoid structure, there is then a disintegration of the dust cells which acts as an irritant, producing a lymphoid hyper plasia, also the fibro-blastic phenomena which characterizes silicosis. Silicotic nodule is now the next stage and this is formed by concentric deposits of fibroblasts occurring at the periphery around pigmented cells.

Infective silicosis implies in practically all cases superimposed tuberculosis. In the main, our discussion will be confined to this angle as the tubercle bacillus is by far the most important organism which modifies silicosis. In the infective type there is a coalescence of the lymphoid islets, foci of caseation appear, which is not present in simple silicosis, and the nodules in the lung take on a greyish appearance—quite a contrast to the blackish nodules of the simple type.

We are most prone to encounter the silicotic problem in the following occupations: Heading the list, of course, is mining, and this will comprise gold, silver, lead, zinc, copper and the anthracite and bituminous coal mining. (2) Quarrying. (3) Stone finishers. (4) Pottery workers. (5) Glass workers, also those workers which engage in spray coating and construction work, such as railways, highways, etc. Of course there are many subdivisions under the above headings, but I feel it is unnecessary to go more into detail.

*History of the Case*

The typical history of the average case is that it begins as "dust fibrosis" and ends as "dust phthisis." Without dust there would be no silicosis and without the siliceous particles the predisposition of tuberculosis would not be present and silicosis would not be the gravely disabling disease that we now know.

It begins very insidiously and is practically always progressive, but during the early years of its progression it is not incapacitating and the individual enjoys his normal state of health. After the presilicotic stage has been passed, and the severity of the process increases, then we attain symptomatology which lessens physical activity.

If the individual affected is the deep-chested, broad-shouldered type of person, the progress of the disease is considerably slower and his tolerance is much greater than the phthinoid type with the flat chest. In the latter the infective process makes inroads a great deal more rapidly, and having less respiratory and constitutional reserve does not stand it so well. We should think of silicosis only as a disease in which the latent tuberculous element plays a large part. The after history of these cases is quite significant. The outlook of the individual is mainly dependent upon his ability to keep the in-

fective agent "bottled up" and inactive. Or, on the other hand, if it becomes active or an infection occurs from without the lung, it may spell exitus. According to Fraser, the actuarial adviser of the Union government of South Africa, the duration of life of the silicotic individual is 13.66 years. The anti-primary stage usually lasts about four years. Primary to secondary stage continues for about four and one-half years. The secondary stage until finality is reached usually comprises from five to six years. These figures indicate very clearly the inherent progressive tendency in most cases of silicosis. In the white individual, longevity should be appreciably greater, and we must recall these statistics are taken from a group of men, the majority of whom are South African natives, whose resistance in combating tuberculosis is very low indeed. Seventy per cent of all deaths in silicosis can be attributed to tuberculosis.

*General Findings of Simple Silicosis*

There are certain cases which finally reach death and no evidence of tuberculosis can be ascertained, and it is assumed that these cases are purely the result of simple silicosis.

The usual symptom, clinically, of silicosis in its earliest detectable stage is the dry, irritating, non-productive cough, worse in the morning and occasionally accompanied by vomiting. Respiratory disability is practically negligible at this time. There is a marked reduction air entry. There is also a characteristic alteration in the normal vesicular type of breathing, to a harsher type due to the predominance of scar tissue. The silicotic chest in the absence of a complicating infection is practically always a dry chest, and an aid to diagnosis is not what you find, but the absence of findings.

In the beginning, the apical zones are less affected than other portions of the lungs, but toward the later stages the lung in its entirety is involved. In an early silicotic stage when the indurative phenomona become a bit marked, it shows as an enlargement and mottling of the hilar shadow, thickening of the trunk shadows proceeding from the hilus to the border of the lung, and considerable arborization, representing a huge network. With the development of the disease the mottling will spread over the lung, oftentimes in a symmetrical manner. Occurring at this time there is a loss of elasticity in the lung, decrease in vital capacity, and dyspnoea may be present.

Breath sounds in the apices are greatly harshened, through the huge ciciatrization, and the lower portions of the lungs show an emphysema. Expectoration is often absent, and rales may only be occasionally found, and this feature makes it a difficult problem, and again portrays the value of Roentgen ray. Normal temperature is often a concomitant feature, even though exitus is only around the corner. Cardiac insufficiency with stasis of the pulmonary and general circulation is the end result.

*Tuberculo-Silicosis*

Grafting tuberculosis upon a silicotic base we find then a coalesence of the fibrotic nodules, a preference for the apical zones, and asymmetry of the thickening processes, cavernous destruction, and a small vertical heart. Clinically, the appearance of all the toxic symptoms of tuberculosis, increase of rales, and also acceleration in the sedimentation test. In certain mines simple silicosis will rapidly pass to the infective type, especially if wet methods have been adopted, and the miners are working in high temperature and relatively high humidity. This naturally predisposes to the development of pathogenic micro-organisms.

I doubt the possibility that such a disease exists as simple silicosis. Realizing that the enormous percentage of deaths in these cases are due to tuberculosis, we are undoubtedly dealing with a condition in which a pre-tuberculosis base is the predisposing factor. Furthermore, I doubt that silicosis would ever develop without this pre-tuberculous base. Simpson and Strachan (3) have recently taken a number of cases of so-called simple silicosis uncomplicated, and by injecting the contents of the silicotic nodule into the groin

of guinea pigs, found that a large proportion of pigs succumb to tuberculosis. Even in its earliest detectable stage there must exist a low grade or latent form of tuberculous infection, even though a clinical demonstrable basis is not evident.

*Schneeberg Lung Disease*

If you will pardon the digression, there is a disease associated with silicosis which has small practical value, because of its limited occurrence in a localized area, and that is the Schneeberg lung disease. Along the borders of Saxony and Bohemia, cobalt and bismuth are mined and these contain a content of arsenic and radium. Developing upon a pneumoconiotic base, pulmonary carcinoma is met with in one half of all the old miners—a combination of dust fibrosis and cancer of the lungs. Ore containing a content of radium may be productive of pulmonary cancer and workers should bear this in mind.

*Radiographic Appearance*

A radiograph of high quality gives us the most reliable single diagnostic criterion. A typical case of moderate degree silicosis will show ofttimes symmetrical mottling of the lung fields, and when this does occur it is almost specific. The first definite evidence of silicosis from the radiograph is the increased fibrosis extending outward from the hilum of the lung, and more or less resembles the leafless tree; as mottling evidences itself, the tree takes on leaves, and this is represented by arborization.

*Regulative Measures*

1. Regulative measures as advocated at present by most mining engineers:
(1) Adequate standardization of ventilation in mines which produce dust phthisis;
(2) Regulate the method and times of blasting.

In choosing applicants for this type of work an initial examination is absolutely necessary, and the flat-chested phthinoid type should be eliminated.

Periodic examination should be given every six months and this should include not only a physical but a radiograph as well.

*Treatment*

Unfortunately, the rehabilitation program has been far from satisfactory. As soon as the presilicotic stage has been diagnosed, the men have been removed from underground work, but remembering the progressive tendency of this disease, this is not sufficient. It is my contention that all of these cases, whether they are in the presilicotic stage, primary stage, or later, they should all be hospitalized and given a period of treatment comparable to tuberculosis since the pretuberculous base must not be lost sight of. In my own work, which is largely referred, I am not fortunate enough to get early cases, and a majority of mine are the advanced tuberculo-silicotic individuals.

To recapitulate allow me to state:

(1) Silicosis apparently has an anatomical basis, as it develops on the site occupied by lymphoid tissue.

(2) First evidence of silicosis is bronchiolitis.

(3) Silicosis is a fibro-blastic phenomenon followed by a dense fibrosis of a nodular type.

(4) Massive fibrosis may develop in an uncomplicated silicosis.

(5) Infection modifies silicosis in the direction of excessive fibrosis.

(6) Tuberculosis is the main infective factor.

BIBLIOGRAPHY

1. A. Mavrogordato. Aetiology of Silicosis (Dust-phthisis), 1930.
2 & 3. Strachan and Simpson. A preliminary study of the pathology of Silicosis as seen on the Witwatersrand.

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**Warfare and Plagues**

WARFARE, LIKE PLAGUES, is intermittent. There may be decades, or even centuries, practically devoid of calamity; in the case of tuberculosis, though there may be cycles, the devastation is never ended. From the dawn of history and beyond, the *white plague* has consistently and unremittingly fulfilled its destructive destiny. Hippocrates, in four hundred B.C., described tuberculosis as the most consistently destructive disease.

C. M. H.

# Immunological Response to Different Proteins of the Tubercle Bacillus

BY
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THE VIOLENT skin reaction produced by extremely small doses of the proteins and protein derivatives, which are the principal active components of Old Tuberculin, is proof that powerful immunological forces are at work here. It is the more surprising to learn that this same protein cannot cause the formation of antibodies, nor can it make normal individuals hypersensitive. Intact dead bacilli, on the other hand, can be the cause of hypersensitiveness.

Some have sought the reason for this difference between tubercle bacilli and tubercle protein in the time factor. A considerable amount of ingenuity has been spent on devices to imitate the slow diffusion of tubercle protein supposed to occur around the tubercle bacillus, but without causing hypersensitiveness. **

Others believe that the anatomical tubercle plays an important part in the production of hypersensitiveness. While it is true that we never find hypersensitiveness without tubercle, we often find tubercle without hypersensitiveness, e. g., in the case of foreign body tubercle. It is possible that contact between a tubercle and tubercle protein is necessary for the production of hypersensitiveness. All efforts to accomplish this contact by introducing tubercle protein into foreign body tubercles by various means, were unsuccessful in producing hypersensitiveness.

A third possibility is the presence in the tubercle bacillus of an unknown substance which is the cause of hypersensitiveness. What do we know of the nature of this hypothetical compound? In the first place, we know that it must be insoluble in water, alcohol and ether. After a hundred extractions with each of these solvents we obtain a powder that is still very active in producing hypersensitiveness. In the second place, we know that it is destroyed by boiling with dilute acids, by treatment with alkali or by prolonged grinding in a ball mill. In the third place, we know that soluble tubercle protein and carbohydrates are the products of its hydrolysis. After prolonged grinding we observe decomposition in soluble proteins and a polysaccharide. This is the specific polysaccharide which has been isolated from the tubercle bacillus on numerous occasions, and which precipitates antiserum in a dilution of 1/100,000.‡

We now obtain the following picture of the active part of the tubercle bacillus. It constitutes about 70 percent of the intact bacillus, and is an insoluble combination of water-soluble tubercle protein (the active part of O. T.) and specific polysaccharide. Each of the two components is a partial antigen. The soluble protein can provoke a skin reaction in a hypersensitive individual, but cannot itself cause hypersensitiveness. The polysaccharide can precipitate a suitable antiserum, but cannot itself cause the formation of antibodies. The combination of these two partial antigens forms the complete antigen, which can provoke a skin reaction, but also causes hypersensitiveness; it can precipitate antiserum but it also stimulates the formation of antibodies.

If we inject an emulsion of the powder obtained by successively grinding and extracting tubercle bacilli into experimental animals, we obtain a serum that agglutinates defatted tubercle bacilli in dilution 1/200 and also gives a precipitate with the specific polysaccharide. Animals injected with any of the numerous kinds of tubercle protein show no formation of agglutinin. After injection of dead bacilli

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**A slight degree of hypersensitiveness is sometimes produced by the use of enormous doses of soluble protein.

‡Boissevain, American Review of Tuberculosis 1935, Vol. XXXI, 547.

(Continued to page 22)

# Resistance and Tuberculosis

BY
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WHEN THE tubercle bacillus invades the respiratory tract in children a primary lesion or Ghon tubercle results. This primary lesion consists of a small circumscribed pneumonic tuberculous process, which may be located anywhere in the lung fields, but is most commonly found in the lower lobes. This Ghon tubercle is the primary lung focus. Next, an associated lymphangitis develops and then the adjacent tracheo-bronchial glands become involved.

The Ghon tubercle tends to spontaneously disappear completely, or become calcified as do the lymphatic glands. Chest signs and symptoms are usually absent, and the primary infection is rarely detected clinically. The bacilli are not walled off as securely as was formerly thought in the calcified tubercle. By absorption of the calcium, the bacilli are occasionally liberated into the blood stream, producing miliary tuberculosis. This primary infection, or Ghon tubercle, causes the body to become acutely sensitized to further infection with tubercle bacilli. This acute sensitization to a protein resulting in inflammation is known as allergy.

Hayfever, asthma and migraine headaches are almost synonymous with allergy. In hayfever, it is usually a sensitization to the pollen protein of plants; in hives, to foodstuffs, such as eggs and strawberries; in asthma and migraine to the protein of many kinds of substances.

The tuberculin reaction is caused by allergy. The Von Pirquet, Mantoux and other tuberculin tests give a positive reaction when a primary infection or Ghon tubercle is present.

There come periods in the individual's life when allergy or the acquired tissue resistance and hyper-sensitivity is lost temporarily. At this time Von Pirquet, Mantoux and other tuberculin tests react negatively. This loss of allergy, or anergy, as it is called, is brought on by overwork, puberty, pregnancy, the menopause, measles, whooping cough, and other conditions which cause bodily reserve depletion. Anergy has been given as the cause for the high mortality rate of adolescent girls, but the most recent work seems to prove this observation wrong, and anergy may prove to be very desirable in an individual. A positive tuberculin reaction points to infection and the degree of reaction is usually a measure of the severity of the infection. This allergic reaction of the body to the tubercle bacillus, brought about by the primary infection, is now thought to be detrimental rather than desirable. Avoidance of infection is again the most desirable, and the most recent evidence adduced shows that the lack of infection does not predispose to the acute fulminating type of lesion, "galloping consumption." In fact, just the opposite prevails, and allergy is now considered a dangerous element in tuberculosis.

Immunity may be described as a condition which causes certain individuals to escape from tuberculosis even though exposed.

This immunity may be congenital, or due to individual or hereditary peculiarities, or it may be acquired—that is, changes produced in the system by a previous attack of tuberculosis, or by vaccination, or inoculation, as in the B.C.G procedure.

Resistance is the ability to ward off, and overcome, the encroachment and effects of the tubercle bacilli. Recovery from tuberculosis depends upon the natural resistance and not immunity. The tuberculin reaction is caused by allergy and gives no measure of immunity.

While it is true that one may exhibit a resistance to a disease and yet be immune to it, in tuberculosis common usage has made the terms immunity and resistance practically synonymous. They are used to express the ability of the individual to fight and overcome the tubercle

bacillus. Resistance differs with each individual.

During the past twenty-five years the subject of resistance and immunity has received much attention. The literature on this subject is voluminous, with the result that a confusion has arisen through which it is difficult to see light. Just what it is that is active in killing the bacilli in the body, or rendering them harmless is not yet known. We do know however, that resistance is not a man-made thing. We either have it, or we don't have it. It is true that immunity to tuberculosis may be acquired when we become infected with tubercle bacilli, but we have not yet learned how to give a greater amount of resistance to one who has not enough to win his fight for life against tuberculosis.

We attempt to increase resistance by one of three methods:

1. By injecting virulent tubercle bacilli.
2. By injecting avirulent tubercle bacilli.
3. By injecting dead bacilli.

It is generally admitted that the use of *virulent* tubercle bacilli is too risky, and that the injection of tuberculin or *dead bacilli* does not give adequate protection. At present, the injection of the killed human bacilli is again being experimented with at Johns Hopkins, and Saranac Lake, and the evidence so far concurs in the belief that the injection of dead bacilli is not satisfactory. Although Soper reports better results with heat-killed S bacilli than with B.C.G.

An attempt to produce immunity by using *avirulent* bacilli is receiving the most attention at the present time and a great deal of work has been done with Bacilli-Calmette-Guerin (bovine tubercle bacilli, isolated from a heifer in 1905 by Calmette, and attenuated by 16 years' culture on a bile potato medium.) The vaccination of infants with this organism began in 1921. This treatment originally consisted of giving approximately three hundred millions of highly attenuated living tubercle bacilli to new-born babies, in the milk feeding, on the third, fifth, and seventh day after birth. At this time the intestine of the new-born is extremely permeable for microbic germs. The advocates of this form of treatment claim that such vaccination protects those in a tuberculous environment from graver infections.

This procedure has been employed so extensively that the injection of B.C.G. bacilli into infants exposed to tuberculous infection can not be considered harmful. That it is beneficial and more helpful than any other agent has not yet been proven.

There is first the questionable safety of prophylatic immunization against tuberculosis by the use of a living organism. A repetition of the unfortunate Lubeck disaster lies within the realm of possibility. The organisms may again become pathogenic. Second, it is claimed by some, that the safer use of dead tubercle bacilli can accomplish as much as Bacilli-Calmette-Guerin. Dr. Calmette of Paris originated this procedure, and France has given its endorsement through the prestige of the Pasteur Institute.

It is impossible in this short article to deal with the tremendous amount of literature already existing. The progress and development of this subject, however, must receive the unbiased attention of all interested in the prevention of tuberculosis. Myers claims that our dangers from tuberculosis really begin when allergy makes its appearance, and they last as long as allergy is present. We can not plant cultures of tubercle bacilli in the bodies of infants without producing allergy.

The value and the limitations of B.C.G. are still to be determined, and it is too early to give an accurate appraisal of this method. We do know that recovery from tuberculosis depends upon the natural resistance and not immunity. Just what it is that comprises resistance, we do not know.

We know that the tuberculin reaction is caused by allergy and gives no measure of immunity; that allergy is dangerous and undesirable; that personal disaster as malnutrition, disease, over work and over

play, all lower the resistance of the natural barriers to infection.

So, until we are given a direct method of *creating* resistance, our efforts in the sanatorium treatment of tuberculosis are directed towards *conserving* and *expending judiciously* whatever resistance the patient is fortunate enough to possess.

PAPWORTH VILLAGE SETTLEMENT; ITS HISTORY AND AIMS
(Continued from page 9)

ply preventive measures long before that resistance has failed? We wait, now, until the invading bacilli have produced symptoms serious enough not only to alarm us, but to alarm our patients, for it is only after the layman detects disease that his physician is called in. This fact, obvious though it be, is often forgotten; but it should be remembered that again and again, especially in tuberculosis, physicians fail simply because they do not see the case in time. We are, as a profession, suffering because we are dependent upon lay diagnosis of symptoms. Can we not alter that? Can we not study the beginnings of disease, as we are now trying to do at Papworth, so that we may gain knowledge of the first signs of disequilibrium and disfunction? I believe that, if once we gain that knowledge, we shall revolutionize the practice of medicine.

Now we say to our lay brethren "Come to us, and we will cure you, if we know how." Before long I hope we may be able to say "Come to us, and we will *keep you well.*"

INTRAPULMONARY INFECTION OF BRONCHOGENIC ORIGIN . . . . (Continued from page 11)

The question very naturally arises as to what the mechanical factors are which produce cross-infection and make the middle of the opposite lung the site of predilection. Consider for instance the left lung. The site of predilection is in continuous line with the direction of the left main bronchus; it is, therefore, reasonable to believe that material aspirated into this region follows the path of least resistance beyond the termination of the left main bronchus, as illustrated on figure 6. When lipiodol was injected through drainage tubes in right chest wall into an old empyema cavity while patient was lying on his left side, the lipiodol reached the main right bronchus through a bronchial fistula, gravitated around the bronchial bifurcation into the left main bronchus, and then out toward the periphery opposite the second to fourth ribs, the site of predilection. The corollary is, therefore, that this part of the left lung is the site of predilection, because the bronchi supplying it are the most direct continuation of the left main bronchus.

This secondary lesion is almost always peripherally situated and involves the parenchyma of the lungs. The infectious material must, therefore, be aspirated into the air vesicles proper. Prolonged maintenance of one posture, such as lying upon the left side when right lung is primarily involved, apparently gives opportunity for the material to drain into the parenchyma of the lung, as was demonstrated artificially in case 4, figure 6. Consequently, patients with suppurative or destructive pulmonary lesions should always be instructed about the reason why they should not lie or sleep on the normal side.

### *Conclusions*

Bronchogenic extension of pulmonary infection from one lung into the middle of the opposite lung is quite frequent; therefore, patients with destructive infectious lesions in one lung should be definitely instructed not to rest or sleep on the normal side.

The site of predilection of bronchogenic cross-infection usually develops in the middle of the opposite lung, that part of the lung which is seen between the second and fourth ribs on roentgen films. The site of predilection is apparently in the path of least resistance beyond the termination of the main bronchus.

Estimation of prognosis in any given case of pulmonary infection does not depend entirely upon the progress or retrogression of the original area of infection, but it is necessary to take into consideration whether or not there is a secondary

infection of other parts of the lungs.

Knowing, therefore, that destructive types of pulmonary infections have a definite tendency to bronchogenic extension and that there is a site of predilection, that part of the lung which is the site of predilection should be studied closely on the roentgen films in order to detect at an early stage the development of secondary infection.

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EARLY PULMONARY TUBERCULOSIS . . . . . (Continued from page 13)

will be able to make more early diagnoses and therefore be able to institute proper measures to effect an arrest of the disease at a time when the prognosis is best. When we reach this Utopian plane in the practice of medicine, we will be able to make some appreciable change in the deaths of young adults caused by tuberculosis.

NOTE: This is first of a series of papers dealing with the management of early tuberculosis. The next, which will appear in an early issue of DISEASES OF THE CHEST, will concern "Collapse Measures in Early Tuberculosis."

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IMMUNOLOGICAL RESPONSE TO DIFFERENT PROTEINS OF THE TUBERCLE BACILLUS
(Continued from page 18)

some agglutinins are formed, but never in a high concentration.

The protein - carbohydrate combination is the active part of the tubercle bacillus and naturally more effective than the bacillus itself which consists partly of inert components as lipoids, soluble protein, etc; in the second place, the intact tubercle bacilli tend to clump and obstruct the pulmonary circulation thus making the use of repeated intravenous injections difficult, while the finely ground protein-polysaccharide complex has almost colloidal size and easily passes through the capillaries; its finely divided particles also expose a much larger surface to immunological attack by the body.

It is interesting to observe that animals immunized by intravenous injections of the protein-polysaccharide complex do not become hypersensitive to tuberculin. To produce hypersensitiveness subcutaneous or intraperitoneal injections are necessary.

When tuberculin is used for diagnostic purposes the use of Old Tuberculin or of the water soluble tubercle protein is clearly unobjectionable. But when tuberculin is used as a theraupeutic or immunizing agent, the protein-polysaccharide complex should be used. If the production of hypersensitiveness must be avoided, intravenous injection is indicated.

*Summary*: A complex molecule containing both tubercle protein and specific polysaccharide causes antibody formation after intravenous injection and hypersensitiveness after subcutaneous injection.

Soluble tubercle protein and specific polysaccharide are partial antigens with hardly a trace of the immunizing power of the parent substance.

# ABSTRACTS

This department is devoted to abstracts of articles carefully and judiciously selected by the Editorial Staff.

HOLMES, FRED G; and RANDOLPH, HOWELL: **Treatment of Lobar Pneumonia by Artificial Pneumothorax.** Annals of Int. Med. Vol. 8 (O.S., Vol. XIII), Number 9. March 1935.

The authors define lobar pneumonia as an infectious (usually pneumococcic) lobar atelectasis of the lung. Referring to the work of Coryllos and Birnbaum, they feel that such a conception of the disease is strengthened.

They described the pneumonic process as follows:

"The pneumococcic infection of the bronchus causes the formation of a very tenacious fibrin-containing sputum which may readily narrow or completely occlude a large bronchus. With the occlusion of the main bronchus to a lobe, lobar atelectasis takes place, beginning at the periphery where the greatest number of alveoli are found, and progressing toward the hilus, while there follows a pneumococcic celulitis proceeding periphreally from the hilus abetted by the negative pressure in the atelectatic area. Physical findings early in pneumonia would seem to substantiate this theory, as we frequently find the breath sounds diminished early in the disease with no definite signs of consolidation. Several Roentgen-rays in our series demonstrate a condition which would seem to be best explained in this way." Holmes and Randolph discuss their series of 18 cases of acute lobar pneumonia treated by pneumothorax and draw the following conclusions.

(1) Artificial pneumothorax causes a marked reduction of pleurisy pain in lobar pneumonia, and it improves the depth of respiration.

(2) It lessens the toxemia.

(3) It seems probable that the duration of pneumonia is shortened, and that at times the crisis is brought about with dramatic suddenness.

(4) In this series less difficulty was encountered from adhesions in adults than in children and there were fewer complications.

(5) The danger of spontaneous collapse and empyema is increased in children.

(6) Total mortality is decreased by use of pneumothorax and the chance of late complications such as abscess, bronchiectasis, or unresolved pneumonia, is probably reduced.

(7) Spreading involvement to new lobes is checked.

MOSES, HENRY W.: **Malignancy in the Lung: Including Eight Primary Carcinomas With Autopsy Findings.** Annals of Int. Med. Vol. 5, No. 6, p. 765.

A study of eighty-one patients with malignant tumors of various types in the lung present the following most common symptoms: Cough, with or without expectoration, which, if present, was usually bloody; to diagnose the condition earlier than is usually done, it is necessary that we concentrate our attention (1) upon those patients who complain of persistent cough without demonstrable causes; (2) upon those patients who expectorate bloody sputum at intervals with no tubercle bacillus in the sputum; (3) upon those patients who complain of general weakness, loss of weight, and do not react to the usual methods of treatment.

At present there is no successful treatment. Surgeons feel that surgery may be of benefit where it is possible to discover the conditions early. Electrical treatment, X-ray and radium may be the means by which this seemingly hopeless problem will be solved.

**HERSHBOECK, FRANK L.: Spontaneous Pneumothorax.** Annals of Int. Medicine, Vol. 4, No. 7, p. 705.

By "spontaneous pneumothorax" Hershboeck means to indicate all pneumothoraces which are not induced by external factors, either accidental or for therapeutic purposes. It occurs most frequently between the ages of 15 to 45; males are affected more often than females, in the proportion of 4 to 1. It may be multilocular or unilocular; partial or total; simple or complicated with effusion, and may be recurrent. Of fifteen bilateral cases reported four have recovered.

Biach of Vienna reported 918 cases of which 715, or 77% were due to pulmonary tuberculosis, 65 to gangrene of the lung, 45 to rupturing of an empyema into the lungs, 32 to injury, various causes of lesser frequency, and 14 of unknown origin.

The author emphasizes particularly the so-called "idiopathic spontaneous pneumothorax" which, judging from reports in medical literature, occurs relatively frequently and in which no definite etiological agent can be uncovered from the history or the examination. These instances arise suddenly, as a rule, with or without diamatic symptoms in the way of pain or local chest discomfort, and with a varying degree of shortness of breath. They tend to run a favorable course, go on to spontaneous recovery in a few weeks with reexpansion of the affected lung and are rarely accompanied by pleural effusion.

The cause of idiopathic spontaneous pneumothorax from a necroptic standpoint is difficult to establish because of the rarety of necropsy findings, but it would appear that rupture of localized emphysematous blebs is the most frequent cause. These blebs may be the result of indurative processes in the subpleural pulmonary tissue, regardless of primary origin; or of emphysematous process in the proximity of adhesions. It is doubtful if pleural adhesions are, per se, a frequent cause. Tuberculosis is probably the most frequent cause of blebs in the apex of the lung. Hershboeck reports five cases of idiopathic origin and several cases with large emphysematous bullae.

**MARIETTE, ERNEST S.: The Dietetic Treatment of Tuberculosis.** Annals of Int. Med Vol. 5, No. 6, p. 793.

In a general discussion of diet as a treatment of tuberculosis the author points out that the over-feeding method of treating tuberculous patients is no longer accepted by the vast majority of specialists. Metabolic studies prove that increasing the diet beyond a certain point is detrimental to the cure of pulmonary tuberculosis because of the increased pulmonary activities necessitated by the increase in metabolism. A diet which is well-balanced and adequate for a man in health is sufficient as a basic diet for a person with tuberculosis unless there is definite gastrointestinal or nutritional disturbance present.

At the Glen Lake Sanitorium a diet of 3000 calories daily is in use. It contains 70 to 100 grams of protein and about 300 grams of carbohydrates. The balance is composed of fat. This includes one quart of milk daily, plenty of raw and cooked vegetables, fruit, meats, etc.

While tuberculosis is distinctly an infectious disease rather than a nutritional one, man is still searching for a diet which will improve the patient's chance for recovery. Thus Sauerbruck aimed to correct the excessive tissue hydration which occurs in tuberculosis by so planning the diet that the sodium chloride content of the urine is reduced to 0.2 to 0.3 grams per day. This is to be done through the substitution of meneralogen, a mixture of inorganic compounds containing 70% of calcium phosphate and lactate for table salt. This diet has apparently been beneficial in lupus but not so beneficial in bone and joint or pulmonary tuberculosis.

All vitamins are considered important in the diet of the healthy as well as the sick. Of particular importance is vitamin D because it has to do with calcium metabolism. However, it is not necessary to give commercially prepared vitamins because we can readily prepare diets from ordinary foods which will contain several times the minimum amount of vitamins required.

BREUER, MILES J.: **Pulmonary Exercise in the Treatment of Tuberculosis.** Annals of Int. Medicine, Vol. 4, No. 4, p. 314.

The author believes that certain "early" cases of tuberculosis which do not respond to the accepted rest treatment, will react favorably to "voluntarily controlled respiration." Such pulmonary exercise is not applicable to cases with advanced or extensive lung involvement, but should be used in certain "low-grade" or "early" cases. As a basis for exercise as a method of treatment Breuer points out that: 1. Pulmonary tuberculosis begins as a small lesion in the right apex where there is least movement on respiration and hence has areation and poorer circulation. Such conditions make a fertile field for infection and spread. 2. Reflex spasm of the muscles of the chest and accessory respiratory muscles tend to further limit circulation of lymph and blood. 3. Because of the limitation of respiratory movement, the patient's supply of fresh air is decreased and general resistance is lowered thereby. 4. Physiologically, pulmonary exercise prevents abdominal stasis through negative pressure in the abdomen during inspiration. The author belives that many digestive and abdominal symptoms seen in the chronic tuberculous individual would be relieved if splanchnic congestion were avoided.

Twenty cases treated by pulmonary exercise are reported. No patient was treated in this manner if the temperature was above 99 F. for more than four hours in the afternoon, or if it ever rose above 100 F.; if there was a productive cough; bacilli in the sputum; coarse rales or signs of lung tissue necrosis. Treatment was begun by directing the patient to take ten deep breaths before each meal at the rate of eight breaths per minute. The frequency of exercise was gradually increased until the breathing was done hourly. Temperature and pulse were carefully watched.

Two of the patients thus treated were dropped because of undue symptoms arising from the exercise. Of the remaining 18, 4 showed no improvement, 14 gained to normal weight, achieved a normal temperature, pulse rate, and blood pressure, and became symptom free.

BLACKFORD, STAIGE D.: **Pulmonary Lesions in Human Tularemia.** Annals of Int. Med. Vol. 5, No. 11, p. 1421.

Although it is well known that tularemia is a blood-born infection, little notice has been taken of the frequency with which it attacks the lungs. Many of the fatal cases of tularemia reported have had an "intercurrent broncho-pneumonia" clinically, or discovered at autopsy. Of thirteen cases of tularemia reported by the author six gave clinical evidence of entrathoracic disease (i.e. pleural effusion 2, bronchopneumonia 2, bronchitis 1, lung abcess 1).

In February 1931, Massee reported finding numerous short gram-negative bacilli, morphologically consistent with B. tularense, in pneumonic areas in the lungs of a patient dying of tularemia. Material from the lung was scratched into the skin of a guinea pig; a granulomatous ulcer developed from which B. tularanse was cultured. There is great similarity of the microscopic pictures seen in the tubercle of tuberculosis and in the caseous necrosis of tularemia. Since the fundamental lesion in both is initially a small area of caseation, this resemblance is not remarkable. It seems peculiar that so little attention has been paid to the similarity of the clinical effects which may be produced by B. tuberculosis and B. tularense. Bronchopneumonia, pleural effusion, lung cavitation, and lung abscess formation are at least some of the clinical conditions which may be caused by both organisms. One wonders if perhaps a few cases of tularemia have not been mistakenly diagnosed as tuberculosis.

Blackford reports a fatal case of tularemia in a negro man, aged 38, who received his infection from an opossum. The clinical diagnosis of pyogenic lung abscess was discredited by microscopic examination of the tissue. The pulmonary damage in this case was extensive and of prime importance as a cause of death.

# QUERIES AND ANSWERS

Careful attention given to all queries. Names will be omitted if so desired.
EDITORIAL OFFICES 1018 Mills Building, El Paso.

*Q. In the issue of May, 1935, of your magazine, DISEASES OF THE CHEST, the leading editorial on "Tuberculin Skin Tests in Children" is of considerable interest in that it advocates dosage of tuberculin which is ten times the usual maximum dose. The amount advocated is a dilution of 1 in 10 which would be 10 mgms. The usual maximum amount as used in the large surveys is 1 mgm.*

A. In the editorial above mentioned we meant to confine our discussion to known contacts. In advocating a dilution of 1 in 10, it was only advised in older children (who are known contacts) and only after a dilution of 1 in 100 had been negative. The 1 in 100 dilution should be used in older children first. In infants the usual 1 in 1000 dilution, of course, would be used. This dilution is about equally sensitive either by the Pirquet method or Mantoux; however, 1 in 100 Mantoux is definitely more sensitive.

*Q. I am especially interested to find out whether there has been any survey conducted which would tend to prove that a dosage of 10 mgs. reveals a larger number of reactors than a dosage of 1 mg. and whether this larger number is worth the extra amount of work involved.*

A. I don't believe that any survey has been made, that is, a mass survey, using a dosage of 10 mgs. And I don't think any one has ever advocated this dose in large surveys. However, men like Gregory, and Weill-Halle of England have used the procedure described in the editorial on known contacts and have always used 1 in 10 dilution in the older children, when a dilution of 1 in 100 was negative. I think they felt that they were justified in the extra amount of work involved, in these older children, who were known contacts.

*Q. I should also be interseted to know whether the author feels that the recommendation of the N. T. A. that the maximum dosage of 1 mg. is suffcient to pick up any reactor to tuberculin of any clinical importance should be revised, and if the author feels that surveys, such as conducted by Meyers, Hetherington, Aaronson and many others, in which a mg. maximum was used would be of more value if the larger dosages were employed.*

A. In the light of certain British observers, a maximum dosage of 1 mg. is not sufficient to pick up all reactors in older children who are definite contacts. For the same reason I feel that the surveys above mentioned would be of more value if the larger dosage were repeated in the older children who were known contacts. It must be remembered that the introduction of tuberculin in any amount into the body of an individual who has never been infected cannot be followed by any ill effects, while the introduction of tuberculin *within the skin* of even a tuberculous patient is not attended by any serious mis-hap. The ill effects that have been reported is due to the subcutaneous introduction and not the intradermal application.

*Q. Who established the first sanatorium for the treatment of tuberculosis?*

A. The first sanatorium was established by H. Brehmer at Goerbersdorf in Silisia in 1859. This institution is still in operation. Brehmer's patients were treated by exercise on the theory that the cause of tuberculosis was a heart too small for the body. Exercise was to strengthen the heart, and help the patient overcome his disease.

## SURGEON ERRANT

**The Life and Writings of William Henry Bucher.**

Dr. Bucher Memorial Committee,
Olive View, Calif.

Exceptional opportunities for service filled the life of William Henry Bucher. Time and again he went on relief expeditions to places stricken by disaster. Volcanoes, earthquakes and floods, famines, plagues and pestilence, warfare, revolution and banditry, all were met by a sympathetic understanding and quiet effectiveness. A veteran warrior against the White Plague, he himself was forced to spend years in "chasing the cure" in the great Southwest. The last fifteen years of his life were devoted to building up the Olive View Sanatorium.

Beside his own biography and the vivid descriptions of his thrilling experiences abroad, his memoirs contain a full and authentic account of the development of Olive View and of the aims and principles underlying its various activities. A group of thought-provoking essays and little gems of verse reveal the deep philosophical nature of the author. Everyone who was fortunate enough to become acquainted with Dr. Bucher, everyone connected with the movement against tuberculosis, everyone who is interested in a stirring tale of travels and adventure, thought and feeling, will enjoy the possession of this book.

**TO OUR READERS!**

**. . . . . Feel free to send us your queries. The Editorial Staff of DISEASES OF THE CHEST will be pleased to give them due consideration.**